

MEAN LUNG CAPACITY UNDER INCREASED O<sub>2</sub> DEMAND

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Translation of "Die Mittelkapazität der Lungen bei erhöhtem  
O<sub>2</sub>-Bedarf," Skandinavisches Archiv für Physiologie,  
Vol. 82, 1939, pp. 201-212

(NASA-TT-F-15598) MEAN LUNG CAPACITY  
UNDER INCREASED O<sub>2</sub> DEMAND (Kanner (Leo)  
Associates) 12 p HC \$4.00 CSCL 06P

N74-30468

Unclas

G3/04 45740

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## STANDARD TITLE PAGE

1. Report No. NASA TT F-15,598		2. Government Accession No.		3. Recipient's Catalog No.	
4. Title and Subtitle MEAN LUNG CAPACITY UNDER INCREASED O <sub>2</sub> DEMAND				5. Report Date August 1974	
				6. Performing Organization Code	
7. Author(s) E. Asmussen and E. H. Christensen, Gymnastic Theory Laboratory and Zoophysiological Laboratory of the University of Copenhagen				8. Performing Organization Report No.	
				10. Work Unit No.	
9. Performing Organization Name and Address Leo Kanner Associates Redwood City, California 94063				11. Contract or Grant No. NASW-2481	
				13. Type of Report and Period Covered Translation	
12. Sponsoring Agency Name and Address National Aeronautics and Space Administration, Washington, D.C. 20546				14. Sponsoring Agency Code	
15. Supplementary Notes  Translation of "Die Mittelkapazität der Lungen bei erhöhtem O <sub>2</sub> -Bedarf," Skandinavisches Archiv für Physiologie, Vol. 82, 1939, pp. 201-212					
16. Abstract The respiration of four subjects exercising on a bicycle ergometer was studied in a steady state both at atmospheric pressure and at reduced pressure and compared with respiration at rest. In contrast to the opinions of several earlier researchers, no general increase in mean capacity was found during work. Verzár's hypothesis of a third form of regulation thus appears to be incorrect.					
17. Key Words (Selected by Author(s))				18. Distribution Statement	
19. Security Classif. (of this report) Unclassified		20. Security Classif. (of this page) Unclassified		21. No. of Pages 12	
				22. Price	

## MEAN LUNG CAPACITY UNDER INCREASED O<sub>2</sub> DEMAND

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According to Bohr (1907), "it has generally been found that /201\* any factor which sufficiently raises the demands placed on the respiratory functions of the lungs also increases mean capacity, and respiration takes place with the lungs filled to a greater degree; conversely, mean lung capacity decreases when respiratory work decreases." Krogh and Lindhard's (1913) experimental data do not agree with this: "We are unable to agree with Bohr's conclusions. We have observed cases where no increase whatever in mean capacity takes place even during heavy work and others showing probably a comparatively slight increase. In one case only have we seen a sudden and considerable increase (0.7 l) take place just at the beginning of work. We believe from what we have seen that the biological importance of variations in mean capacity is very slight; but we propose to put off the discussion until more material shall have accumulated."

More recently, Verzár (1933) has again taken up the question, and he summarizes the principal result of his article, "The regulation of lung volume," as follows: "...In addition to the acceleration and deepening of respiration, there is also a third form of respiratory regulation: in enlargement of lung volume, particularly important under an increased demand for O<sub>2</sub>."

Since the experimental material published so far is very meager and some of it, in our opinion, is hardly suitable to serve as the basis for a discussion on the functional significance of a /202

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\* Numbers in the margin indicated pagination in the foreign text.

possible change in mean lung capacity as an important component of respiratory regulation under an increased  $O_2$  demand, we considered it to be of use to carry out the studies presented here.

If a change in mean capacity is to be interpreted as an important part of respiratory regulation, we would have to expect such regulation to be particularly clearly manifested in work, particularly in the steady state, in which  $O_2$  demand and ventilation are matched to one another through a change in depth and frequency. Thus we have made all determinations in a steady state at normal pressure or, in several cases, at a pressure of 495 mm Hg.

The transition from rest to work (Verzár) or the recovery period after the cessation of work (Bohr) appears to us to be unsuitable for such determinations, since the transition can very easily produce considerable changes in mean capacity, in purely mechanical terms, if the rest and work positions are not exactly identical; these changes can of course not be interpreted as regulatory effects (for example, see the studies by Grosse-Brockhoff, Schoedel and Springorum (1936)).

### Method and Results

The experiments were performed on subjects in good physical condition: three males, E.A., H.A. and E.H.C., and one female, E.L.L. Lung volumes were determined with a Krogh spirometer, and residual capacity was determined by the  $H_2$  method (see Lindhard (1925)). According to Verzár (1933), one receives the impression that the  $H_2$  method is relatively unsuitable for residual capacity determinations, since the author apparently attributes the problems associated with a determination of injurious<sup>1</sup> area to residual capacity determinations. Verzár prefers a

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[Translator's note: sic; authors may have meant "damaged."]

plethysmographic method in which the total volume of the body is determined. The application of this method to determinations during work is very limited, however, since the extra heat produced will greatly influence the air volume of the plethysmograph even during extremely little work and thus makes a reliable determination of body volume impossible. Verzář's plethysmograph must have an air volume of about 450 liters, and even for the very small amount of work involved, which means the additional production of about 1 cal per min (a lung ventilation rate of about 16 liters per min), work time may definitely not exceed 203 1 min if the quantity of heat produced is not to make the volume measurement totally worthless (1 cal transferred quantitatively to the 450 l would mean a volume increase of more than 10 l!).

For the exercise machine we used Krogh's bicycle ergometer, and all determinations both at rest and while working were made in the working position on the ergometer. It appeared important to us to use leg work for such experiments, since arm work can more easily affect thoracic position mechanically by immobilization. The subject breathed through a zero-resistance valve system, and the expired air could be directed either into a Douglas bag or a Krogh spirometer by turning a three-way valve. All tube connections were quite wide, and the spirometer was very well balanced, so no appreciable resistance could affect either inspiration or expiration. Respiration frequency was recorded with a Marey tambour. The three-way valve was operated without the subject's being able to notice anything; he had his eyes closed throughout the entire trial. Mean depth was calculated for given ventilation with the aid of expiration volume and breathing frequency. A number of expirations were also recorded with the spirometer as a control; while the subject was connected to the spirometer, he was asked -- after expiration had begun -- to exhale completely so that the amount of reserve air could be determined. After a brief period of normal respiration, vital capacity and sometimes also residual capacity were determined.

On each day of testing, between 180 and 1440 kgm/min work was studied in the case of the male subjects; for the female subjects, the work varied between 90 and 900 kgm/min. In addition, trials in the partial-vacuum chamber were performed with subject E.A. and E.H.C.

The values for lung ventilation are plotted versus intensity of work in Fig. 1<sup>2</sup>.

Figs. 2 through 5 contain the values for lung volume on the various experiment dates. The mean position of the lungs is indicated by the sum of reserve air and half the depth of respiration. Mean capacity is greater by an amount equal to residual capacity. All determinations made showed unchanged residual capacity up to the highest intensities of work. For example, we obtained the values shown in Table 1 with subject E.A. /204

TABLE I. SUBJECT E.A.: LUNG VENTILATION AND RESIDUAL CAPACITY

Lung ventilation in l/min	5	20	40 <sup>1</sup>	60	80
Residual capacity in l	1.20	1.24	1.28	1.37	1.25

We are certainly justified in assuming that residual capacity is practically unchanged for the ventilation values studied here, so the mean position plotted in Figs. 2 through 5 is also a reliable indication of mean capacity. The vital capacities of the four subjects vary considerably: Female subject E.L.L. has a vital capacity of about 3.50 l; E.A. and E.H.C. have about 5.0 l, while H.A. has about 8.0. A feature common to all is that vital capacity remains constant, except for the highest level of work, in which case a slight decrease occurs. Since residual capacity

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<sup>2</sup> All volumes are given for room temperature and the pressure shown.

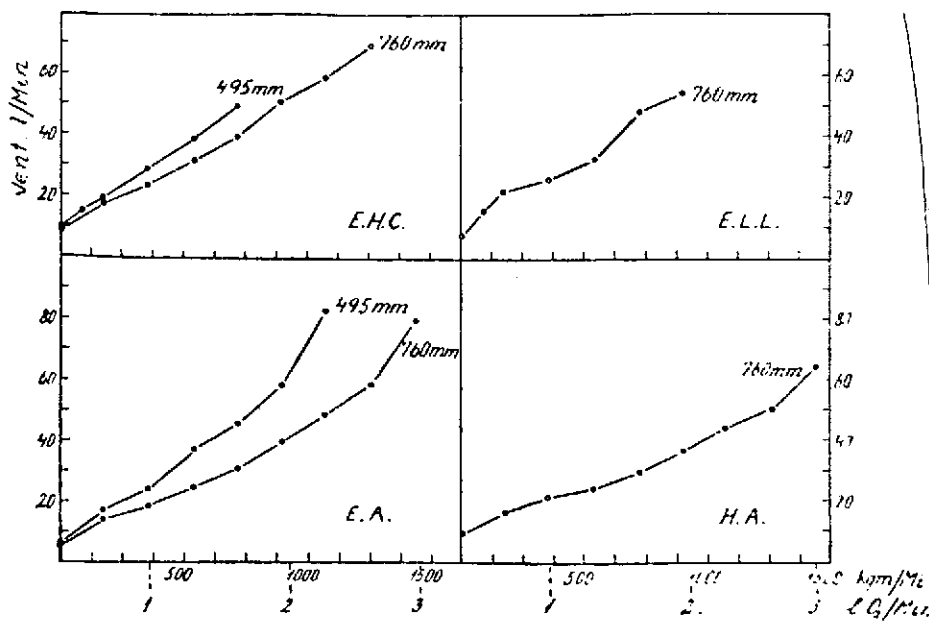


Fig. 1. Lung ventilation for different intensities of work and levels of O<sub>2</sub> uptake.

likewise remains practically constant, the total capacity of the lungs has remained almost constant up to maximum work. The observed decreases can certainly be attributed to mechanical factors in immobilization of the thorax.

It can also be seen from the curves that mean capacity is constant or decreases slightly with increasing work intensity in the case of subject E.L.L., is constant or increases slightly in the case of E.A., and increases slightly in the case of E.H.C. but markedly in the case of H.A. The rise in the case of H.A. is by no means regular, however. For example, we find a constant or reduced mean capacity for ventilation levels up to 40 l/min in the lower graph, and a distinct rise only after this, whereas the upper graph exhibits a rise even for ventilation levels of about 20 l/min. /205 /207

The readings taken on E.A. and E.H.C. in the partial-vacuum chamber exhibit no differences from the normal trials, either with regard to vital capacity or to mean capacity (Figs. 2 and 4, lower graphs).

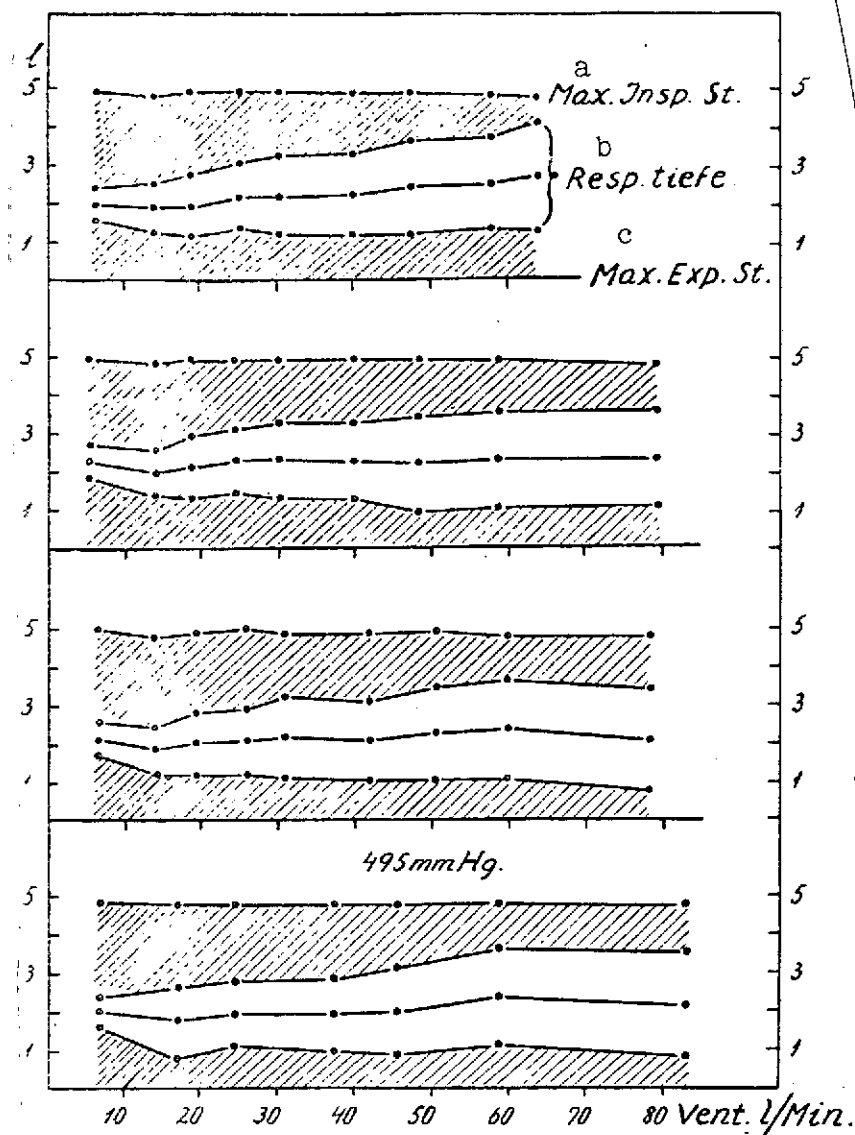


Fig. 2. Subject E.A.: vital capacity, complementary air; depth of respiratory and reserve air, at pressures of 760 mm Hg and 495 mm Hg.

Key: a. Position of maximum inspiration  
 b. Depth of respiration  
 c. Position of maximum expiration

Complementary air and reserve air must become smaller and smaller with increasing depth of respiration, in such a manner that the decrease effects both volumes to approximately the same extent in the cases of E.A., E.H.C. and E.L.L., while in the case



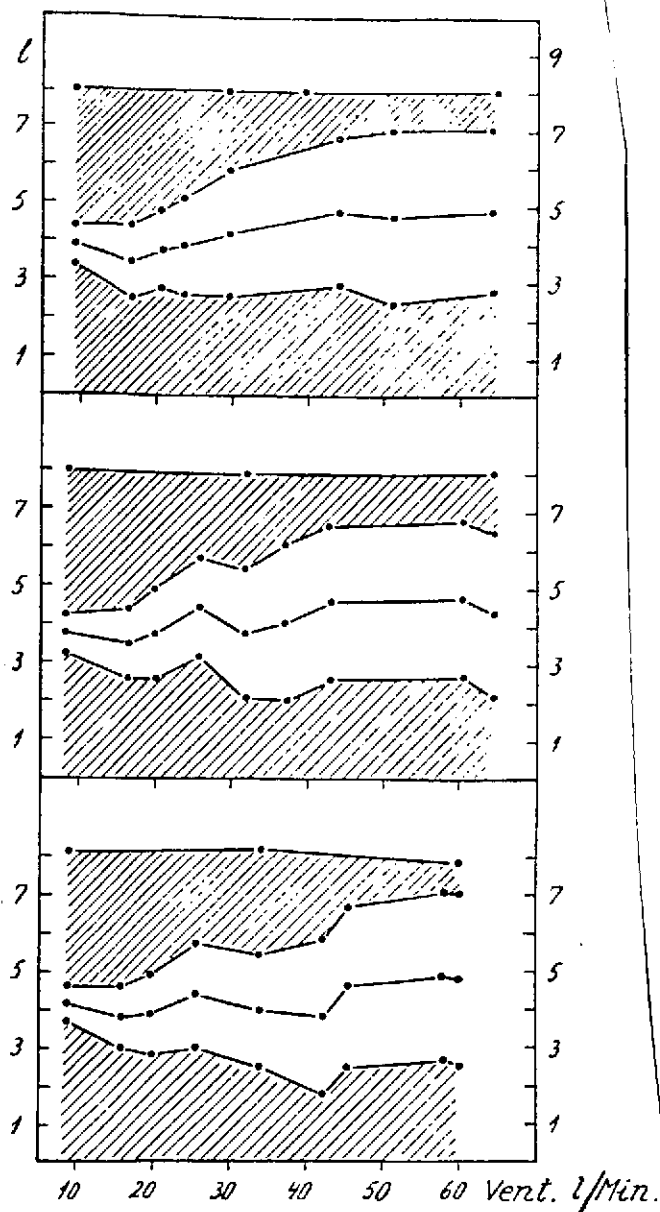


Fig. 3. Subject H.A.; cf. Fig. 2 for explanation.

of H.A., the increase in depth /208 of respiration actually occurs at the expense of complementary air.

Bohr found a distinct drop in vital capacity immediately after a run, in one case from 3.31 to 2.21 l (1 min after the run) and a corresponding increase in mean capacity. We measured vital capacity under a work load of 1440 kgm/min ( $O_2$  uptake about 3.0 l/min, ventilation about 80 l/min) in a definite steady state (subject E.A.) and then redetermined vital capacity after the cessation of work (in unchanged position!) at short time intervals. Fig. 6 shows the results; it can be seen from this that vital capacity is practically constant and of the same magnitude as we normally find it in E.A. at rest.

In other experiments, Bohr likewise observed an increase in mean capacity if the subject moved from a lying to a standing position. Bohr relates this difference to metabolic differences in the applicable positions; he writes: "The observed decreases in mean capacity appear to be quite naturally related to this; in /209 the lying position, the lungs are adapted to the lower level of

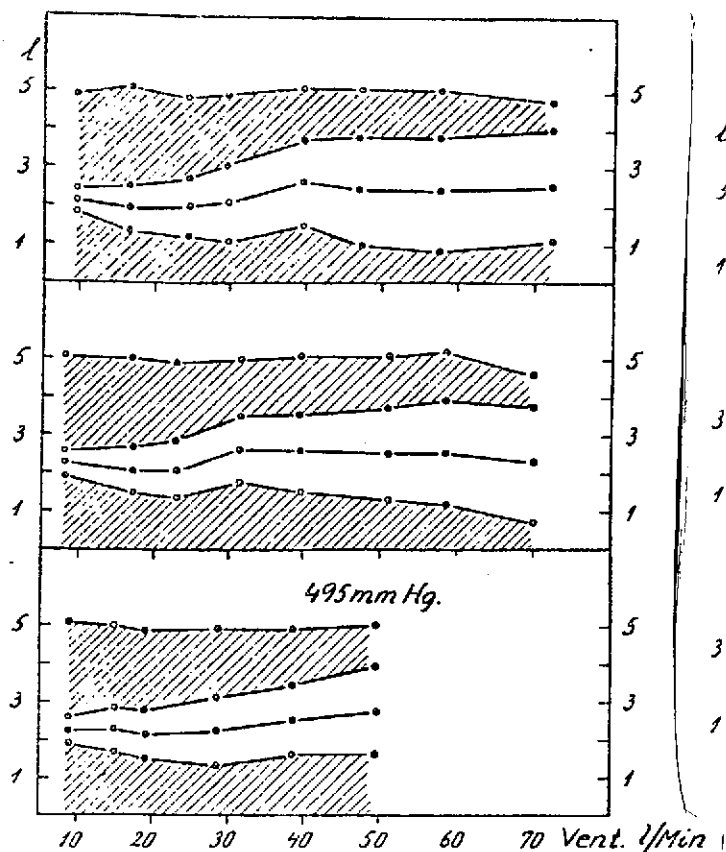


Fig. 4. Subject E.H.C.;  
cf. Fig. 2 for explanation.

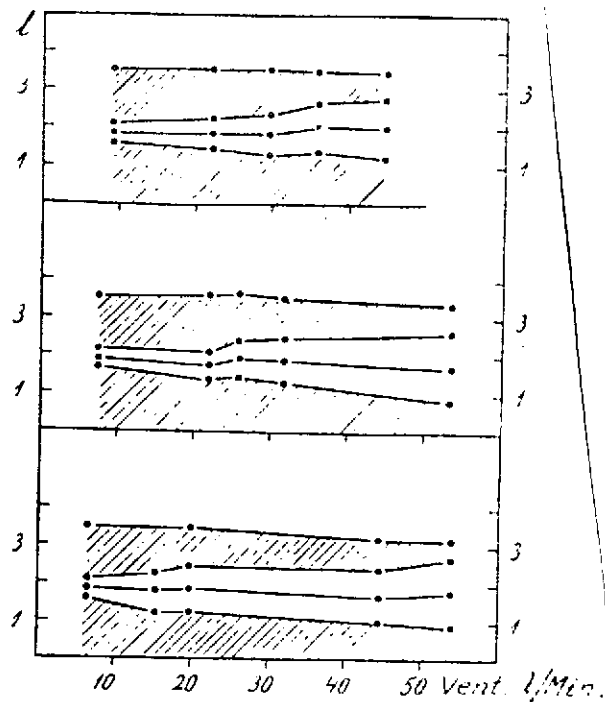


Fig. 5. Subject E.L.L.  
(female); cf. Fig. 2 for  
explanation.

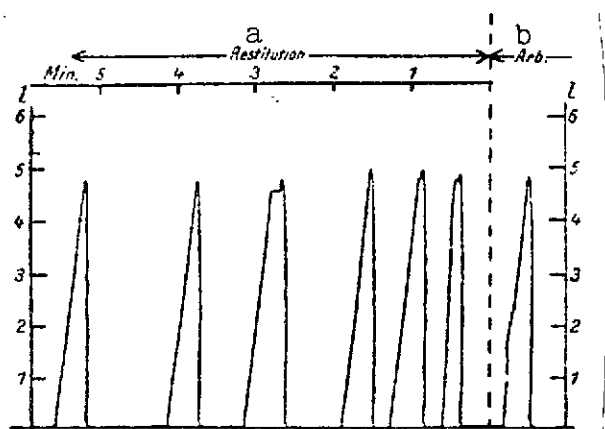


Fig. 6. Subject E.A.: vital  
capacity during and immediately  
after 1440 kgm/min work.

Key: a. Recovery; b. Work

metabolism through their more  
collapsed state and the resul-  
tant small surface area."

In experiments described  
in a previous study (Asmussen,  
E.H. Christensen and  
T. Sjöstrand), we were able to  
show that the change in vital  
capacity and mean capacity at  
a constant metabolic rate is  
a function of variation in the  
amount of blood filling the  
thorax. Fig. 7 shows lung

volume both for different positions (head high,  $+60^\circ$ , and head low,  $-60^\circ$ ) and with different amounts of blood filling the lower extremities and the thorax. It can be seen from Fig. 7 that appreciable changes in means capacity and total capacity can be caused by shifts in the blood. The changes in lung volume which Bohr observed during changes in position were probably caused by purely mechanical factors or by blood shifts and thus cannot be interpreted as regulatory effects. /210

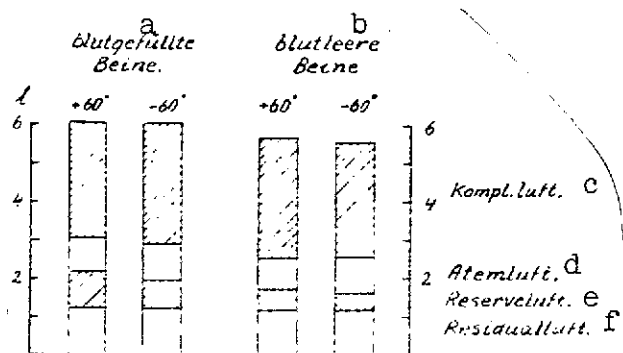


Fig. 7. Subject E.A.: lung volumes associated with position changes and altered peripheral blood distribution.

Key: a. Legs filled with blood; b. Legs emptied of blood; c. Complementary air; d. Tidal air; e. Reserve air; f. Residual air

### Discussion

According to Verzár, who took up Bohr's theories again, a regulatory increase in lung volume should occur as a "third form of respiratory regulation" under an increased  $O_2$  demand, as well as under conditions in which  $O_2$  uptake is made difficult, e.g. under low  $O_2$  pressure. We would then have to predict that such a third form of regulation would be particularly conspicuous during work in

a steady state in which the two other forms of regulation (acceleration and deepening of respiration) are subject to appreciable loads. For methodological reasons, Verzár could only study very small and brief levels of work, and his results suffer from the fact that an initial change in mean capacity can perhaps be interpreted exclusively as the result of changed body or arm position during work. During work in a steady state, increase in mean capacity is not such a regular phenomenon that it can be looked upon as a

regulatory measure. Krogh and Lindhard (1913) found, as we have now, that large individual differences exist: an increase is found in the case of one subject, while no increase or even a reduction in mean capacity is found in the case of another. Of our four subjects, the one (E.L.L.) who has the smallest vital capacity (3.5 l) and thus certainly would have been able to use a third form of regulation if available exhibited no increase at all in mean capacity, not even at the maximum work level for her of 900 kgm/min. In contrast, H.A., with a very high vital capacity of 8.0 l, exhibited a distinct increase in mean capacity at the same work level and an  $O_2$  uptake rate of about 2 l/min during one trial, although this work was very far from the maximum for him. Rather, it appears as if the high vital capacity of this subject allowed him to establish the depth of respiration randomly between very wide limits.

At reduced  $O_2$  pressure, it is likewise not possible to demonstrate a regulatory increase in mean capacity during trials at rest. Hasselbalch (1912), who indeed first confirmed Bohr's findings (1908), was able to show in subsequent experiments that the changes in mean capacity which occur during the inhalation of /211 low-oxygen air (10%  $O_2$ ) vary individually just as much as during work. Hasselbalch summarizes his work as follows: "The independent physiological significance of mean capacity for chemical respiratory regulation can thus not be very great."

Not even during work in low-oxygen air could a regulatory increase in mean capacity be reliably detected, as Figs. 2 and 4 show. One subject, E.A., showed no changes up to ventilation rates of 45 l/min; the other, E.H.C., showed a moderate increase in capacity, but this was no greater than under normal pressure. The variations which occur during changes of position have already been discussed and provide no points of reference for assuming a "third form of regulation."

We believe that we may conclude from the above that no definite increase in mean capacity -- at least on the order of magnitude found by Verzář -- occurs in the subjects studied either during work or at reduced  $O_2$  pressure. We therefore believe, in agreement with Hasselbalch and Krogh and Lindhard, that a regulatory adaptation of mean capacity to  $O_2$  demand does not occur.

### Summary

It is possible to show that a definite increase in mean capacity is found neither during work in a steady state nor at reduced  $O_2$  pressure at rest or during work. A "third form of respiratory regulation" (Verzář) thus does not appear to occur in the four subjects tested by us at ventilation rates between 5 and 80 l/min.

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